The Journal of HEREDITY

PINHEAD PINUPS

(SEE PAGE 211)

The Inheritance of Birthmarks

A Lethal-Spasm Gene in Cattle

Hair-Color Chemistry in Negro Albinos

Utilization of Technical Personnel in Total War

A Check-List of Lethal Genes in Livestock

VOLUME 35 JULY, 1944 NUMBER 7

The American Genetic Association

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COLOR DIFFERENCES DUE TO A SINGLE GENE

Figure 1

The Strong twins, whose parents are both normally pigmented negroes, demonstrate extreme range in differences in color due to a single gene. As studies reported elsewhere in this issue show, the differences in pigment formation appear to be due to differences in oxydation of melanin precursors. In white subjects the albino gene is said to affect the dimensions of the hair, but this does not appear to be so in negroes (see page 201). Other photos of the Strong twins on page 218.

HEREDITARY CONGENITAL LETHAL SPASMS IN JERSEY CATTLE

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In September 1942 a commercial dairyman of California brought to our laboratory a living grade Jersey male calf afflicted with a congenital type of spasm that always resulted in death within a few days or weeks after birth. The first anomalous calf had appeared in his herd about five years before. Although he had not been alarmed by a few other sporadic cases occurring within the next two or three years, the larger percentage of such calves born more recently caused him to consult a local veterinarian. The veterinarian could make no definite diagnosis. After checking the nutritional regimen, he became convinced that the defect could not be attributed to faulty nutrition, and suggested sending an affected specimen to the Cutter Laboratory at Oakland, California, for autopsy. All the tests made there for an infectious agent were negative. Since the laboratory report suggested that the condition might be hereditary, the dairyman decided to bring a defective calf to us. The information obtained at this interview strongly indicated that the defect might be hereditary. Two of us (Gregory and Mead) accordingly visited the dairyman to obtain all the available data from his herd and to examine at first hand the breeding records.

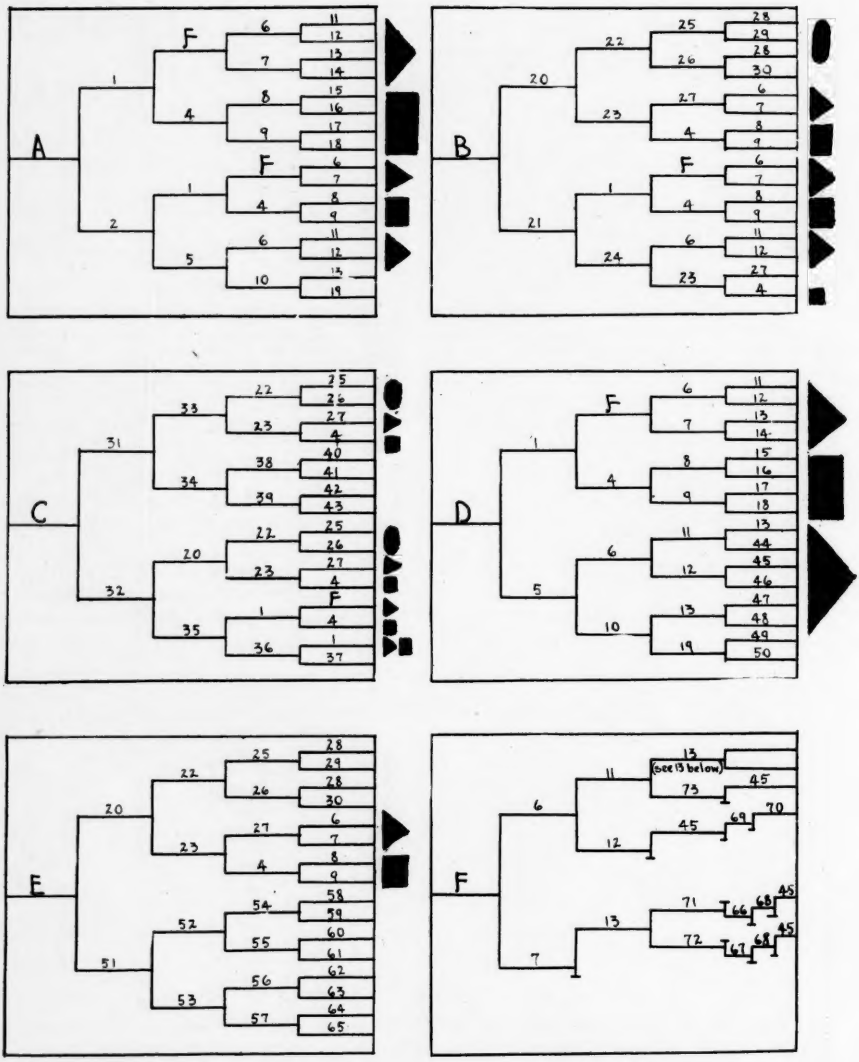
The defective calf was left at our laboratory. We planned to observe it for a few days before making an autopsy and preparing certain tissues for histological study. The specimen, six days old when brought to us, had been allowed to nurse daily. It appeared robust and normal except for certain convulsive movements. While lying down it manifested continual intermittent spasms of the head and neck, primarily in a vertical plane. The notes taken at the time fail to mention uncontrolled movements that may have occurred when the head was resting on the ground. The calf could stand, but never did so voluntarily. On its feet it was very uncertain and unsteady because of intermittent spasms in all legs. The legs appeared normal in size and shape, but in standing or walking were placed wide apart, apparently to facilitate balancing. The spasms of the head and neck appeared more violent during standing or walking, perhaps because magnified by the spasms of the legs. A sudden noise or shock neither initiated nor intensified the spasms.

While in our possession the calf had a good appetite and drank milk greedily, but with extreme difficulty because of the convulsive movements of the head and neck.

Upon the morning of the third day in our laboratory the calf developed an extreme case of diarrhea. It died sometime during the evening two days later, at eleven days of age. An autopsy was not possible. This was the only spasmodic calf the authors had an opportunity to observe. Our notes on it agree well with the dairyman's description of a dozen or more specimens. The dairyman stated that a few days before death the head may become drawn to one side or back, as in a tonic type of spasm. Other dairymen have mentioned this symptom.

Evidence of Inheritance

The dairyman kept fairly complete records and knew his herd thoroughly, as he and his family did all the work directly connected with it. What work he did not do himself he supervised very closely. His methods of feeding and management conformed to the sound practices generally employed in that locality.



RELATED PEDIGREES INVOLVING THE SPASMODIC GENE

Figure 2

Each animal in all the pedigrees is coded by a letter or number. The different shaped blocks to the right of the pedigrees identify three lines of ancestry which recur in the pedigrees. Sires *A, B, C,* and *D* have been proven to be heterozygous by progeny tests. A sufficient number of test matings has been made to prove Sire *E* to be homozygous normal. Pedigree *F* is presented to bring out the interrelationship of Sire 45 to other animals not shown in pedigrees *A, B, C, D,* and *E*. A combination of progeny tests and pedigree analyses indicates that Sires 45, 68, 69, and 70 are heterozygous. It is significant that Sire *F* appears in the pedigrees of all of the heterozygous animals. Sire 45 appears four times in the pedigree of the sire of Sire *F*, and twice in the pedigree of his dam.

The herd consisted of about 70 Jersey cows, none of which was registered. Its average yearly fat production was about 420 pounds. For the past 11 years the owner had been using closely related registered sires secured in a neighboring state from one of the highest-producing Jersey herds of the country. The first bull so purchased was born in 1931 (Figure 2, bull *A*) and remained in service 5½ years. His daughters were uniformly high producers. According to the dairyman, occasional spasmodic calves began to appear when this sire was mated to his own daughters. In 1934 a second bull (Figure 2, bull *B*) was purchased. He was related to bull *A* and also proved to sire high production. When he was bred to all the daughters of bull *A* then in the herd, calves afflicted with spasms began to appear with greater regularity.

In 1940 the dairyman secured from the same out-of-state breeder a third bull (Figure 2, *C*), which was related to the first two. By this time all the cows in the herd were daughters of the first two bulls. The first calves sired by bull *C* began to appear about January 1, 1942. A complete record of all calves born from then until October 8, 1942, (the date of the authors' visit), was available. With the exception of a few sired by bull *B*, all calves born between these dates were sired by *C*. Six spasmodic calves, four females and two males, were observed (Table I). The first resulted from mating bull *B* to his own daughter. The other five were offspring of *C* from daughters of *B*. During this period bulls *B* and *C* also sired forty-seven normal calves. (It should be remembered that all the cows in the herd were daughters of bulls *A* and *B*.) Thus six calves out of a total of 53, or approximately one-eighth, were spasmodic. If one autosomal recessive gene causes the spasmodic condition and both bulls *A* and *B* are heterozygous, one would expect 6.6 spasmodic calves. The records gave ample proof that bull *A* was heterozygous. A cow named Buttercup sired by him produced a spasmodic calf when bred to bull *B*. Evidently, then, the lethal spasmodic anomaly is conditioned by one autosomal recessive gene.

Before the three heterozygous bulls were introduced, the frequency of this defective gene is assumed to have been 0. After a succession of three heterozygous sires had been used for 12 years, the frequency of this defective gene in the present generation approached .2929, the theoretical limit.

This dairyman is now using a fourth related bull, which is under test for the spasmodic gene, being bred to daughters of bulls *A* and *B*.

Through the assistance of the local Agricultural Extension Service, arrangements were made to visit a second dairyman, a breeder of registered Jerseys who for some years had used a bull (Figure 2, *D*) closely related to those used by the first dairyman. According to this breeder, five spasmodic calves had been born in his herd a few years earlier (Table II). These represent about one-eighth of all calves born from the mating of sire *D* to his own daughters. One cow produced two defective calves, a male and a female. These results also indicate that one autosomal recessive gene conditions the defect, and agree with observations in the first herd.

Bull *D* was eventually replaced by *E* (Figure 2). Bull *E* was not closely related to *D* and when bred to the latter's daughters proved to be homozygous normal.

The analyses of these two California herds using related sires purchased from the same herd yielded positive evidence that four of the five bulls tested were heterozygous for the gene that conditions congenital lethal spasms. Indica-

TABLE I. Spasmodic Calves Born in the First Herd Studied

Sex	Date born	Sire	Dam's Sire
Female	Feb. 1942	*B*	*B*
Female	July 1942	*C*	*B*
Female*	Aug. 1942	*C*	*B*
Male	Sept. 1942	*C*	*B*
Male†	Sept. 1942	*C*	*B*
Female	Sept. 1942	*C*	*B*

*Sent to Cutter Laboratory for autopsy and culture.
†Observed by authors.

tions point, therefore, to a relatively high frequency of the defective gene in the herd that was furnishing the sires.

Lethals and a Constructive Breeding Program

The first dairyman is confronted with a problem in selecting a future herd sire. Since three heterozygous sires have been used in succession during 12 years, the frequency of the lethal gene approaches the theoretical limit of .2929 in his breeding cows, and the continued use of heterozygous sires would result in a calf loss of about 15 per cent. Since high production is rather well fixed in the family producing the sires, the dairyman dislikes to resort to outcrossing, with the consequent risk of losing production in his herd, even though an outcross would almost certainly stop the appearance of lethal calves. It is impracticable for a dairyman keeping grade cows, even though he uses registered sires, to undertake a breeding program for eliminating lethal or defective genes.

Such a program should be the responsibility of the purebred breeders who derive much of their income from the sale of breeding stock. The elimination of recessive lethals and other hereditary defects should be just as essential a part of a constructive breeder's program as progeny testing and selection for increased production.

The commercial dairyman, on the other hand, can participate in the program. If there is a high frequency of a specific lethal gene, his herd is ideal for testing young bulls for the gene in question. If no specific recessive lethal is suspected in the commercial herd, but one wishes to test a bull for recessive lethal genes in general, sire-daughter matings may be used. A bull under test for recessive lethals can also be proved for his ability to transmit production.

TABLE II. Spasmodic Calves Born in the Second Herd

Sex	Date born	Sire	Dam's Sire
Female	1933	*D*	*D*
Male	1936	*D*	*D*
Male	1936	*D*	*D*
Female	1937	*D*	*D*
Female	1938	*D*	*D*

If such a program were under way for each generation, a constructive breeder could test in commercial herds several promising young bulls. From these he could select a succession of herd sires free from all simple recessive lethal genes and at the same time capable of transmitting high production uniformly. If such a program were followed in the purebred herd for only six generations, the frequency of each specific lethal gene originally present would be reduced to a negligible quantity even though it had originally approached the theoretical limit of .293.

Obviously this program would require close cooperation between the breeder who is attempting to do constructive work and the commercial dairyman who participates in the program. The breeder of purebreds must take the initiative. If the suggested program were carried out on a large scale throughout the country the present frequency of recessive lethal genes among purebred cattle could be greatly reduced.

Origin of the Mutant Gene and Its Distribution

Inquiries have revealed that this defect has occurred in at least two other Jersey herds not previously mentioned. In an adjoining state, spasmodic calves were reported in a herd using the paternal grandson and maternal great-grandson of sire I. This situation was not surprising, since sire 1 figures so prominently in the pedigrees of proved heterozygous bulls *A*, *B*, *C*, and *D* (Figure 2).

At least six defective calves were produced in another California herd. The first was sired by a son of bull *D* from a dam descended from sires 27 and 68 (Figure 2, *B*, *D*, and *F*). The next spasmodic calf was sired by a second son of *D* from a dam that traces to 6, 7, and 68 (Figure 2, *A*, *D*, and *F*). The third calf was sired by the second son of *D* from a dam descended five different ways from sire 68. The fourth calf implicates sire 68 even more. Its sire is descended from 68 two different ways, and its dam eight different ways. Even though this

type of pedigree analysis is less dependable for tracing genes and determining genotype than direct progeny tests, it strongly supports the conclusion that sire 68 is heterozygous. Judging from an analysis of pedigrees *A*, *D*, and *F*, (Figure 2), sire 45 need not be heterozygous if the mutation occurred in 68 or if 68 received the gene from his dam. The fifth calf apparently settles this point. Its sire is the second son of *D*; and the only connection of its dam to the mutant stock is through sire 70, the paternal grandsire of 45 (Figure 2 *F*). Evidence strongly supports the conclusion that sires 45, 69, and 70 are also heterozygous. Although cow 4 appears in all the pedigrees of heterozygous sires *A*, *B*, *C*, and *D*, she is not descended from any of the heterozygous bulls and should be considered homozygous normal.

Several generations intervene between the older animals (45, 68, 69, and 70), which were assumed to be heterozygous by indirect progeny tests coupled with pedigree studies, and the younger animals (especially *A*,*B*, *C*, and *D*), which were proved to be heterozygous by progeny tests alone. If sires 45 and 68 are heterozygous, all the intervening animals descended from them are potential heterozygotes; but we have no data that prove the genotypes of intervening animals 1, 2, F, 5, 6, 7, 10, 11, 12, 13, and others, although 1 and F are probably heterozygous (Figure 2 *A*, *B*,*C*,*D*,*F*). Many of the cows and their descendants have outstanding production records, and many of the sires uniformly transmit exceedingly high production. Since this stock is in great demand, the spasmodic gene is being widely disseminated. Spasmodic calves may therefore appear in many herds throughout the United States, and the identity of sires 45 and 68 may be of assistance to any one tracing the causative gene. Sire 45 is Melia Ann's Golden Wolseley, Reg. No. 62598: and sire 68 is St. Mawes, Reg. No. 72053. These two sires will appear only far back in the pedigree of their descendants now being born.

The high frequency of the spasmodic gene materially reduces the value of this stock. High production, on the other hand, is apparently well fixed and this compensates in part for the disadvantage of the lethal. It is to be regretted that breeders of such valuable stock are reluctant to use a simple routine progeny test, so familiar to geneticists, for eliminating such lethals from valuable seed stock.

Relation of Spasms to Other Defects

Numerous examples of hereditary nervous disorders have been reported in mammals. This spasmodic mutant is compared with other somewhat similar mutants that have occurred in rodents and domesticated mammals. It is certainly not homologous with the "waltzing" type of defect observed in mice,[4] rats,[1] and rabbits.[2] Though the word "shaker" describes it well, it differs from the three nonlethal shaker mutations reported in mice. (See Snell[6] for original references.)

There are several papers concerning a nervous disorder of goats. According to Lush[5] the defect is conditioned by heredity, though the exact manner of inheritance has not been determined. Because of differences in behavior alone, the spasmodic condition of calves cannot be regarded as identical with the nonlethal hereditary nervous disorder of goats which is induced by sudden noise or shock.

Superficially, at least, the spasms in calves have more characteristics in common with congenital palsy of guinea pigs reported by Cole and Ibsen[3] than with any of the other defects mentioned. Both defects are congenital, lethal, and recessive; but here the resemblance ends. Without being stimulated, affected calves exhibit continual intermittent spasmodic movements; and sudden noises or shocks neither initiate nor noticeably intensify the spasms. The defect is probably not homologous with any previously reported in rodents or other mammals.

The authors are indebted to Mr. Enoch Torpen, of the Agricultural Extension Service, for aid in obtaining some of the data and to the dairymen in

whose herds the spasmodic defect was studied, for their splendid cooperation and interest.

Summary

A new type of congenital hereditary spasm in Jerseys is described. Afflicted calves exhibit continual intermittent spasmodic movements of the head and neck, usually in a vertical plane. When the animals are forced to stand, spasms in both front and hind legs hamper standing or walking. Noise and shocks neither initiate nor intensify the spasms. Aside from the spasms, such calves up to a few days of age appear to be normal and vigorous and to have good appetites. They die, however, within a few weeks after birth.

The spasmodic condition is inherited as a recessive autosomal lethal. All the herds in which defective calves appeared were using closely related bulls purchased or descended from a single herd of high-producing purebred Jerseys. The defect was uncovered by sire-daughter matings and by using a succession of related heterozygous sires. Four out of five sires' progeny tested for the defect in two herds proved to be heterozygous. Evidence indicates that this gene has been present in the stock for 10 or more generations. The dairymen who used three heterozygous sires in succession during almost 12 years increased the frequency of the defective gene in his herd to approximately the theoretical limit. A similar condition had probably been reached in the purebred Jersey herd from which the heterozygous sires were purchased.

This lethal is probably not homologous with any other hereditary lethal so far reported in rodents or other mammals.

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FRATERNAL TWINS AND ERYTHROBLASTOSIS

(The Rh Gene Again)

Double Ova Pregnancy in Which Rh + Twin Developed Erythroblastosis. — Potter (*Jour. Pediatrics* 24:371) reports that in May 1943 twins were born at the Chicago Lying-In Hospital to a normal woman aged 32 who had had one uneventful pregnancy nine years previously. The twins were males, the placentas and amniotic sacs were separate. One infant survived and was entirely normal; the other died of erythroblastosis on the second day of life. The mother has shown no anti Rh agglutinins but is OMN Rh—; the father, ON Rh+; the 9 year old daughter, O Rh+; the living infant, OMN Rh—; the dead infant, OMN Rh+. It is concluded that the father was heterozygous for the Rh factor and that two ova were fertilized, one by a sperm carrying on Rh+ gene and the other by a sperm carrying an Rh— gene. The Rh— mother had been sensitized to the Rh factor either in the previous pregnancy, when an Rh+ fetus had been carried, or possibly during the course of this pregnancy by the Rh+ twin. The antibodies produced in the mother passed through the placentas of both fetuses but affected only one which was Rh+. The cells of the Rh+ fetus contained the Rh antigen and could be agglutinated by the Rh antibodies; the cells of the Rh— fetus contained no antigen and therefore could not be agglutinated. The fetus whose cells were agglutinated developed erythroblastosis; the one whose cells were not agglutinated remained well. — *Jour. Amer. Med. Assoc.* 125:813. 1944.

HAIR OF NEGRO ALBINOS

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THE macroscopic color of negro albino head hair varies from white to yellow and reddish according to previous authors.* Microscopic studies of negro albino hair have been made of two cases by Frédéric;[6] one case by Bloch;[5] and eleven cases by Pearson, Nettleship, and Usher.[11] Herewith is reported a study of the head hair of eight additional negro albinos coming from four families. The pedigrees, general descriptions, and pictures of these albinos were reported earlier by the authors.[12] Albinism is inherited as a dominant in the *M* family; as an irregular dominant, in the *J* family; and as a recessive in the *B* and *W* families. An albino mother and four albino children occur in the *M* family, whereas hair samples were obtained from one albino only in each of the other three families. The designations refer to the pedigrees published by the authors.[12]

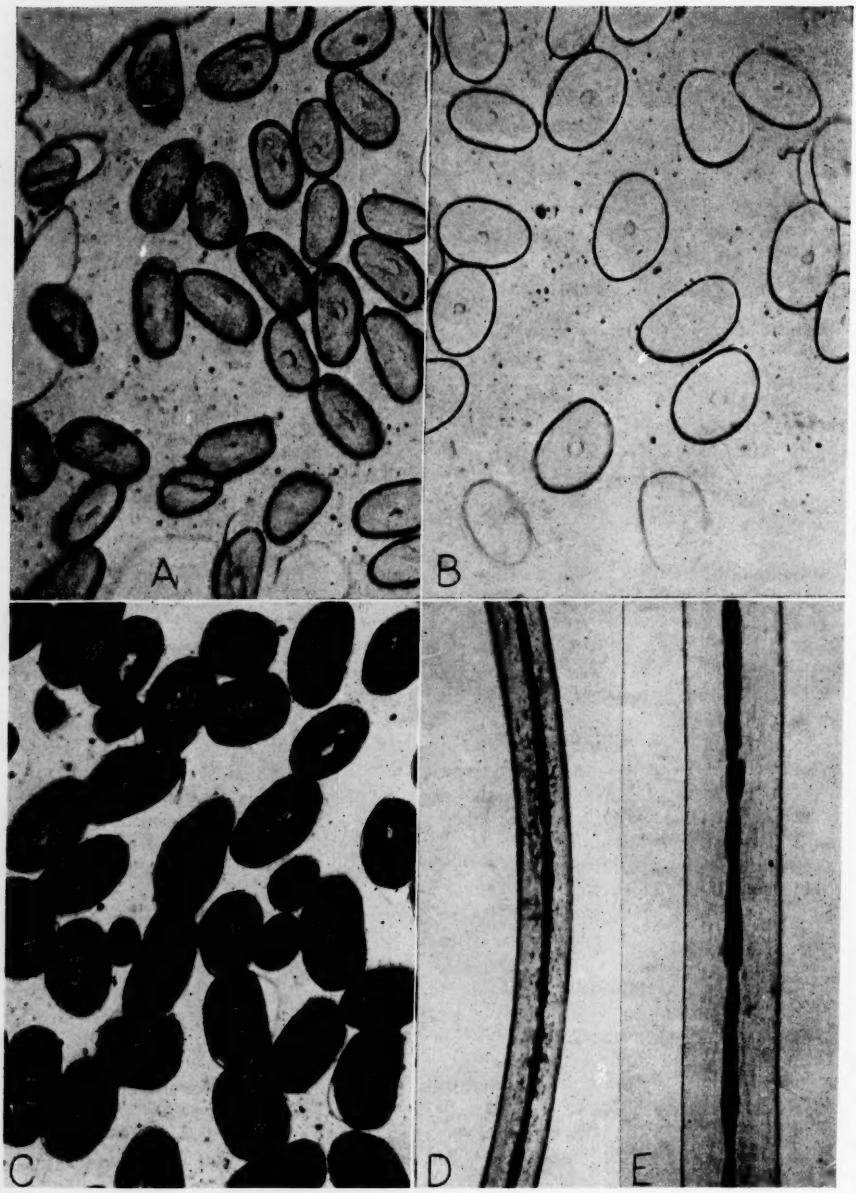
From each negro albino a lock of hair was collected from the tip of hair growing at the back of the head. With one exception, *J* V-1, whose hair had been artificially straightened, the hair of the negro albinos was typically kinky. The macroscopic hair color was uniform over the entire head in each case. The authors owe an exact determination of the macroscopic color of the hair to Mrs. Sarah Brooks Martin who used Maerz and Paul's *Dictionary of Color*.[9] The hair samples were first washed in carbon tetrachloride and then matched with the correct color plate under bright natural light (but not sunlight). The color determinations together with age, sex, and type of inheritance of albinism are given in Table I.

The macroscopic albino hair colors are not "pure." Their location, with one exception, on Maerz and Paul's plate 12 indicates a certain amount of grayness. Yellow to orange colors begin on plate 9 as pure colors; successive plates are overlaid with gray up to plate 16. Adjacent colors such as E3 and F3 are closer to the same color than E3 and F4 (one above the other). Thus, of the negro albinos, *M* IV-5, *M* IV-3, *M* IV-1, *M* IV-4, and *B* III-4 have the pale straw and yellow colors; *M* III-3, *W* IV-17, and *J* V-1 have the more reddish hair colors.

For microscopic analysis of the negro albino hair, whole mounts and cross sections were used. The hair was cleaned in carbon tetrachloride, dried, and mounted in diaphane or balsam as whole mounts. For cross sections, a tuft of about 200 hairs was assembled and sectioned according to Hardy's method.[7] The sections were then mounted in diaphane or balsam. Whole mounts only were made of the hair of *M* IV-1 and *M* IV-5, small boys, whose hair was too short for cutting sections. Figures 3*A* and *B* show cross sections of the hair of the albinos *J* V-1 and *B* III-4. Figure 3 *D* and *E* show whole mounts of the hair of the albinos *M* IV-1 and *B* III-4. Finally in Figure 3*C* appear the cross sections of the hair of a normal dark negro woman, Lola S. The microphotographs are all ×150.

Microscopic examination of the cross sections of the hair of six albinos shows a very small amount of granular pigment present in the cortex of the hair of *B* III-4, *J* V-1, and *W* IV-17. No granular pigment was detected in the hair of *M* III-3, *M* IV-3, and *M* IV-4. None of the negro albinos studied by Pearson, Nettleship, and Usher had any pigment granules in the hair, although these authors found a small amount of granular pigment in certain white race albinos. Bloch found no granular pigment in the hair of the one negro albino studied by

*Frédéric,[6] Atgier;[2] Blaschko;[4] Pearson, Nettleship, and Usher;[11] Musser;[10] Wakefield and Dellenger;[16] and McCrackin.[8]



HAIR OF NEGRO ALBINOS

Figure 3

Cross sections of the hair of two Negro albinos (*J* V-1 and *B* III-4) are shown at *A* and *B*. No pigment granules are visible—compare with sections of normal Negro hair at *C*. *D* and *E* show mounts of the hair of albinos *M* IV-1 and *B* III-4. Cortical air spaces appear in the hair of *B* III-4, but are not present in the hair of *M* IV-1. Medullary air spaces were present in some hairs of all samples studied.

him, though Frédéric reported sparsely distributed pigment granules in the hair of both of his two negro albino cases. In *B* III-4, *J* V-1, and *W* IV-17, a few scattered pigment granules were observed in the cortex of the hair in cross sections; also a few pigment granules were seen leaking out of the freshly cut sections. Presumably the latter granules came from intercellular spaces of the cortex where pigment granules are abundantly located in normal hair. The few pigment granules present can not be distinguished in the photographs, although they may be detected by focusing with the fine adjustment of the microscope. Such a small amount of dark granular pigment present could have little effect upon the general hair color. Obviously the macroscopic hair color is determined by the amount of diffuse reddish-yellow pigment present. The macroscopic color determinations of Table I therefore indicate the relative amounts of reddish-yellow diffuse pigment present in the hair of the eight albinos. The reddish-yellow diffuse pigment is uniformly distributed over the cortex of the hair in each case. In cross section the hair of *J* V-1 appears a pale honey color; *W* IV-17, amber; *M* III-3, *M* IV-3, *M* IV-4, and *B* III-4 appear pale straw.

Air spaces are present in the medulla of some hairs of all negro albinos studied. Not all hairs possess a medulla, and the medulla is frequently interrupted, as in normal, non-albino hair. Medullary air spaces appear in Figure 3*D* and *E* as long black areas in the central region of the hair. The medullary air space is continuous in the portion of the hair of *M* IV-1 shown in Figure 3*D*. In Figure 3*E* showing hair of *B* III-4 three different medullary air spaces occur, between which extends the medullary tissue, clearly differentiated from the cortex. Medullary air spaces were present to an appreciably greater extent in the hair of the albino children, *M* IV-1, *M* IV-3, *M* IV-4, and *M* IV-5, than in any other albinos.

When present, air spaces in the cortex of negro albino hair occur as minute, irregular, longish droplets, appearing yellowish to brownish when viewed with transmitted light and clustered in the central part of the cortex. No cortical air spaces were found in the hair of *J* V-1, *W* IV-17, *M* III-3, and *B* III-4. Air spaces in the cortex were numerous in the hair of the children *M* IV-1, *M* IV-3, *M* IV-4, and *M* IV-5. Figure 3*D* shows the cortical air spaces in the hair of *M* IV-1; Figure 3*E* shows the absence of cortical air spaces of *B* III-4. Medullary and cortical air spaces observed in the negro albino hair appear similar to such air spaces observed in a gray hair sample and a blond hair sample of white race hair studied by the authors. Air spaces, appearing white by reflected light, lighten the appearance of gray or blond hair in which they occur. It is presumed that the medullary air spaces which were more extensive in the *M* family children than in any other albinos, and the cortical air spaces, which occurred only in these children, contributed to the paleness of their hair. Pearson et al[11] describe large medullary air spaces and many small cortical air spaces in negro albino hair.

Pigment Chemistry

The effect of an oxidizing agent upon negro albino hair and upon several control hair samples was studied. Hair of the eight negro albinos, a normal dark negro, and a sample of light brown, red, and very blond hair of the white race were cleaned and put into individual screw top vials of three per cent hydrogen peroxide (H_2O_2) Parke-Davis, (U.S.P.). The vials were kept in the dark. Temporary whole mounts were made and examined under the microscope to ascertain color changes. After four hours of treatment, the hairs of *M* IV-1, *M* IV-3, *M* IV-4, *M* IV-5, and *B* III-4 were pale straw and did not differ preceptibly from the untreated hair of these individuals. The remaining negro albino hair samples of *J* IV-1, *W* IV-17, and *M* III-3 were definitely bleached after four hours in H_2O_2 but still retained some yellow color. Even after 22 hours of treatment, some hairs in each of the latter three samples of

negro albino hair were a pale yellow or straw color whereas other hairs in each of the three samples were oyster white. One hair of the normal dark negro hair was noticeably lighter after treatment with H_2O_2 for four hours, the other hairs of the sample being as dark as the control. After 51 hours the dark negro hair had bleached to a light chocolate color. The three white race hair samples were also bleached by H_2O_2. A preceptible color change occurred in the red and blond hair after only one hour in the oxidizing agent. In two hours some of the red hairs became oyster white, while a faint yellow color was still present in one hair of the sample. The blond hair became white after four days in H_2O_2. The light brown white race hair became pale straw and yellow after four days in the oxidizing agent. The bleaching of hair pigment with an oxidizing agent such as H_2O_2 has been known for a long time. Our results are in complete accord with the extensive experiments of Saller and Maroske[13] on bleaching red hair of the white race with H_2O_2. Our experiments also confirm that of Bloch who found a pale yellow color retained in his negro albino hair sample after treatment with H_2O_2. Since the hair color in Bloch's case was originally a pale yellow, he did not observe a bleaching action of the oxidizing agent.

The effect of a reducing agent, stannous chloride ($SnCl_2$), was next studied. Hair of six female negro albinos, *J* V-1, *W* IV-17, *B* III-4, *M* III-3, *M* IV-3, and *M* IV-4 were placed in individual screw top vials of freshly prepared $SnCl_2$ solution (Merck, U.S.P.). The hair samples of the two young albino boys, *M* IV-1 and *M* IV-5, were not used because the hair lengths were extremely short in comparison with those of the other six albino hair samples. In addition, light brown, red, blond, and gray samples of white race hair were similarly treated with $SnCl_2$. No heat was applied in these experiments except upon the second sample of gray hair in contrast with the method of Saller and Maroske. Temporary whole mounts were made in order to determine under the microscope the extent of the darkening. The darkening of the negro albino hair was preceptible after four hours of treatment. The reduction proceeded at different rates in the different albino hair samples. Furthermore, the reduction proceeded at slightly different rates sometimes in different regions of the same hair. The action of $SnCl_2$ upon two different samples of one hair each from the six negro albinos are described in Table II. The gray hair and light brown hair of the white race were unchanged by remaining in $SnCl_2$ without the application of heat. However, the authors obtained a yellowing of gray hair by heating it in $SnCl_2$, thus confirming the results of Saller and Maroske. The blond hair and red hair samples darkened slightly but not nearly to such a degree as the negro albino hair.

The six albino hair samples that had been treated with $SnCl_2$ were mounted in celluloid, and sections were cut according to the method previously described. The characteristic size and shape of the hair remained the same as before treatment. The cuticle was distinct in all cases. The medulla was sharply defined in five cases, absent in one, *M* IV-3. The medulla is absent completely or in part from all normal, untreated hair. No air spaces were present in hair treated with $SnCl_2$ since all such spaces were filled with liquid. The ground color of the cortex varied with the sample, being in each case a deeper yellow or orange than the respective control.

In treated hair of *J* V-1, the ground color of the cortex varied in intensity in different sections from yellow to rich reddish orange. The color in any one section was uniformly distributed, however. Numerous jet black granules were scattered over the cortex and medulla, being slightly more numerous in the medulla and medial half of the cortex than in the peripheral half of the cortex. The black granules appear similar in size, shape and distribution to the black melanin granules found in normal negro hair. In addition, reddish brown pig-

ment granules were distributed throughout the cortex, but they were more abundant in the peripheral half of the cortex. Two sections were observed that contained only reddish brown granules and no black granules.

The ground color of the cortex of treated hair of *W* IV-17 was orange brownish, deeper in the peripheral half than in the medial half of the cortex. Numerous jet black pigment granules, more abundant than in any other reduced negro albino hair, were scattered over the entire cortex and medulla.

The cortex of the treated hair of *M* III-3 appeared a uniform pale yellow. Neither black nor reddish brown pigment granules were found. A second sample confirmed the absence of pigment granules in the reduced hair of *M* III-3.

In *B* III-4 the peripheral half of the cortex appeared burnt orange; the medial half, straw. Both cortex and medulla contained numerous jet black pigment granules, apparently as many as in *J* V-1.

In the treated hair of *M* IV-3, some sections appeared pale yellow; others, pale orange in ground color. The color of any particular cortex was uniform. A fine sprinkling of black and dark reddish brown pigment granules was distributed over the entire cortex. The medulla was not present in this particular hair.

The ground color of treated *M* IV-4 hair was pale dusty yellow, lighter immediately around the medulla. The medulla and cortex contained both black and reddish brown pigment granules, more abundant than in *M* IV-3, but less abundant than in *J* VI-1, *W* IV-17, and *B* III-4.

Thus the rapidity and degree with which the chromogen in the various negro albino hair samples may be reduced in the absence of heat to reddish yellow diffuse pigment, reddish brown and black granular pigment varies in the six cases investigated. Apparently neither the type of inheritance of the primary albino gene; i.e., whether dominant, irregular dominant, or recessive, nor the age of the individual albino are related to the extent of reduction observed. Furthermore the initial color of the albino hair is not connected with the extent of the reduction since the hair of *B* III-4, originally pale straw, became quickly reduced, as did the originally darker hair of *J* V-1 and *W* IV-17.

Saller and Maroske heated the colorless hair of white race albinos in $SnCl_2$ and obtained a reduction of the chromogen to a yellowish red color which by further reduction became dark. Upon observing whole mounts they found "occasional" pigment granules in the reduced albino hair of the initial $SnCl_2$ treatment. The reduced albino hair was bleached by treatment with H_2O_2. Red hair of the white race was also darkened by heating in $SnCl_2$ according to Saller and Maroske. These authors conclude that the reddish yellow diffuse pigment is derived from black granular pigment by oxidation. The reddish yellow pigment may be oxidized in turn to the colorless condition observed in white race albinos. The various yellow and reddish yellow colors of negro albino hair represent partial oxidation stages of the reddish diffuse pigment, according to Saller and Maroske.

The present authors' results from oxidation and reduction of negro albino hair support Saller and Maroske's conclusions. The presence of reddish brown pigment granules in addition to black pigment granules in the reduced hair of *J* V-1, *M* IV-3, and *M* IV-4 is morphological evidence in favor of the theory that the black granular pigment may be oxidized to give a reddish yellow diffuse pigment. Possibly the reddish granular pigment represents an intermediate stage between black granular pigment and reddish yellow diffuse pigment. The actual proof of this theory will have to await chemical syntheses of the pigments. Arnow,[1] from oxidizing dopa-melanin concludes that the pigment in red human hair is an oxidation product of black melanin. Baker and Andrews present additional evidence, using a similar method, in favor of this view.

Six samples of negro albino hair from *J* V-1, *W* IV-17, *B* III-4, *M* III-3, *M*

IV-3, and *M* IV-4 were put in vials containing 2.5 per cent of potassium hydroxide (KOH) and examined six hours later under the microscope. In all cases the hair became limp, greatly swollen, the edges of the hair fuzzy, and the color faded. No color was present in the treated hair of *M* IV-4; a pale straw color remained in the treated hair of the other five hair samples. Saller and Marokse found the reddish yellow diffuse pigment of white race red hair to be soluble in KOH.

The fineness of white race albino hair compared with normal hair of the white race has been pointed out by a number of previous authors. To determine if this fineness is characteristic of negro albino hair also, measurements of lengthwise and transverse diameters were made of 100 cross sections of six female negro albinos and six normal control negro females. The averages in micra of lengthwise and transverse diameters of 100 cross sections of each hair are given in Table III. The ratios between lengthwise and corresponding transverse diameters for each hair sample are also given.

The lengthwise diameters (maximum diameters)of the cross sections of ten normal dark negroes studied by Steggerda and Seibert are as follows: 90.04 $\pm$ 1.15; 94.17 $\pm$ 1.40; 102.89 $\pm$ 1.49; 95.59 $\pm$ 1.05; 74.91 $\pm$ 1.24, for five males and 80.61 $\pm$ 1.27; 82.01 $\pm$ 1.41; 88.72 $\pm$ 0.98; 105.96 $\pm$ 1.34, for five females. Figure 3*A* and *B* shows the size difference in the hair of *B* III-4, one of the coarsest, and *J* V-1, one of the finest of the albino hair samples.

It is clear from Table III that the range of variation among the measurements of negro albino hair is similar to that found among the six controls and the ten individuals studied by Steggerda and Seibert. Hence unusual fineness of hair such as many authors have described in white race albino hair can not be considered a secondary effect of albinism in negroes.

The authors are indebted to Dr. Allen B. Griffen, of the University of Texas, for making the photographs; to Mrs. Sarah Brooks Martin, of the University of Texas, for macroscopic hair color matching; to Dr. J. T. Patterson, of the University of Texas, for laboratory facilities to the first author for a part of the time during which the study was made; and to Dr. P. J. Thomas, Charity Hospital, New Orleans, Louisiana, for assistance in locating three of the albino families.

Summary

The macroscopic hair colors of the eight negro albinos studied varied from pale straw to yellow to reddish. A very small amount of black granular pigment was present in three of six cases examined microscopically in cross sections. Air spaces were present in the medulla of all negro albinos and also in the cortex of four of them.

The pigment in negro albino hair was bleached with an oxidizing agent, H_2O_2. A reducing agent, $SnCl_2$, on the other hand, darkened six negro albino hair samples studied. Individaul differences in reaction are discussed as well as the chemistry of the observed reactions.

Probably the reddish yellow diffuse pigment is derived from granular pigment by oxidation since this process appears to be reversed when the negro albino hair is treated with a reducing agent.

Unusual fineness of hair is apparently not a secondary effect of albinism in negroes.

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TABLE I. Origin and color classification of albino negro hair used in this study. Designations refer to pedigree numbers in the *Journal of Heredity*.[12]

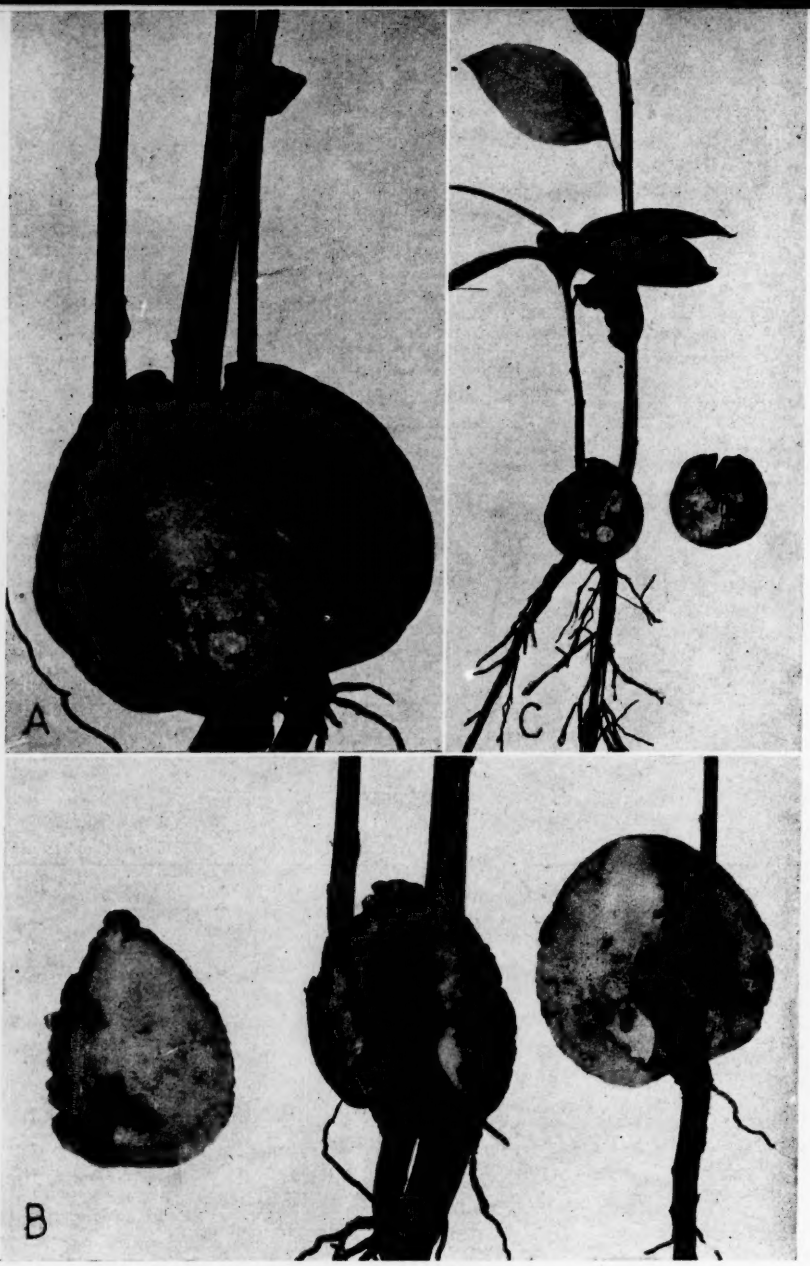
Albino	*M* IV-5 ♂	*M* IV-4 ♀	*M* IV-3 ♀	*M* IV-1 ♂	*M* III-3 ♀	*B* III-4 ♀	*W* IV-17 ♀	*J* V-1 ♀
Color plate	12 D2	12 E4	12 E2	12 E3	12 I5	12 H2	11 I6	12 I6
Age in years	3	4	5	11	29	21	36	27
Type of inheritance	dominant					recessive		irregular dominant

TABLE II. Darkening of negro hair with stannous chloride.

Sample number	Hours in $SnCl_2$	*J* V-1	*W* IV-17	*M* III-3	*B* III-4	*M* IV-3	*M* IV-4
1	4	Deep golden	Canary yellow	Pale yellow	Part of a hair deep golden; rest, golden brown	Golden	Golden
1	44	Golden-brown	Part of a hair canary yellow; part, golden-brown	Part of a hair golden; most yellow	Part of a hair deep golden; part, deep chocolate brown	Golden, like *M* IV-4	Golden; darker than M III-3
2	18 (for all except *J* V-1)	Chocolate brown (36 hours)	Part of a hair pale yellow; part, light chocolate brown	Pale yellow	Deep golden brown	Deep golden brown	Deep golden brown
Control (untreated hair)			Amber (lighter than *J* V-1)	Pale straw	Pale straw	Pale straw	Pale straw

TABLE III. Lengthwise and transverse diameters of cross sections of negro albino hair compared with normal negro hair.

Albino negro	Lengthwise diameter ± S.D.	Transverse diameter ± S.D.	Ratio of lengthwise to transverse diameter	Age in years
W IV-17	90.51 ± 1.26	61.32 ± 0.84	1.48	36
B III-4	105.53 ± 0.95	70.04 ± 0.84	1.50	21
J V-1	85.16 ± 0.11	48.20 ± 0.74	1.77	27
M III-3	71.82 ± 0.84	37.80 ± 0.53	1.90	29
M IV-3	82.32 ± 1.26	41.58 ± 0.63	1.98	5
M IV-4	71.51 ± 1.47	43.58 ± 0.84	1.64	4
Normal negro				
Lola S.	94.50 ± 1.68	62.58 ± 0.84	1.51	42
Smith	101.54 ± 1.26	69.83 ± 0.84	1.45	16
B. Jewel	83.92 ± 1.44	46.29 ± 0.78	1.81	8
Betty	85.91 ± 0.78	54.95 ± 0.67	1.56	20
Mrs. Wilson	80.92 ± 0.89	51.85 ± 0.56	1.56	60
Bessie	119.55 ± 1.67	67.16 ± 1.22	1.78	25



MULTIPLE GROWTH AXES

Figure 4

Normally the avocado seed is composed of two large hemispherical cotyledons. *A* and *B* show two views of a seed which had three cotyledons and three independent growth axes (embryos). *C* shows a seed divided into four cotyledons and with twin growth axes. Some of the plant axes are apparently identical in genetic constitution and others are obviously different. All appear to be gametic in origin, in distinction to the multiple embryos which arise by the budding of maternal nucellar tissue in citrus and mango, essentially a form of asexual reproduction, giving seedlings with the same genetic constitution to the mother plant.

MULTIPLE EMBRYOS IN THE AVOCADO

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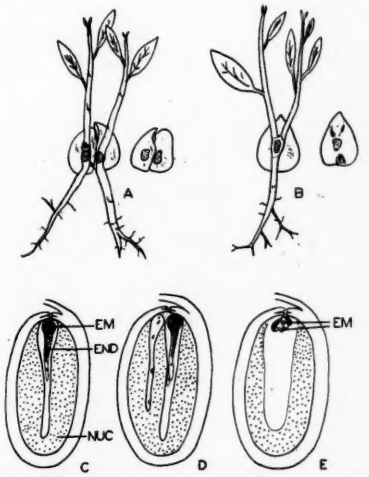
THE avocado (*Persea americana* Miller) generally produces a single plant from one seed. The normal seed is composed of two large, hemispherical, pointed cotyledons joined together by a mass of tissue, the cotyledonary node. It develops following fecundation of the egg cell in the single embryo sac (Figure 5*C*).

Seeds which give rise to two individual plants each are observed occasionally in seed-beds (Figure 5*A*). These should not be confused with branching of a single stem close to the cotyledonary node which occurs frequently in seedlings (Figure 5*B*). The leaf characters of the individual plants may be similar or may vary considerably. Thus one plant may have smooth leaves and the other a rougher or more corrugated leaf surface. In one instance of twin seedlings, one of the plants had new growth which was copper colored, a character generally associated with varieties belonging to the Mexican race, while the other had green tip growth which is common to varieties of the Guatemalan race.

The occurrence of seeds with three cotyledons from which a single plant develops is common while seeds with four or more cotyledons which give rise to polyembryonic plants as described above are much less common. In the latter the multiple cotyledons are closely appressed and are often highly convoluted. By dissection, however, the mass of cotyledons can be separated into pairs each of which gives rise to a single plant.

Another type of multiple embryo consists of a single seed with three cotyledons, giving rise to three distinct plants. The three cotyledons are connected by two cotyledonary nodes, each of which contains a plant axis, and a third axis arises from one of the cotyledons. In this case the cotyledons and plant axes



MULTIPLE EMBRYOS IN THE AVOCADO
Figure 5

A—Polyembryonic avocado seed split to show the two distinct plants and four cotyledons. *B*—Normal seedling with two cotyledons, but branching close to cotyledonary node. This can be mistaken for polyembryony. *C*—Longitudinal section through normal ovule which contains a single endosperm (*END*) and nucellus (*NUC*). *D*—Longitudinal section through abnormal ovule with two embryo sacs one of which contains a young, spherical embryo. *E*—Ovule from young fruitlet containing two small embryos in a single chamber.

are probably identical in genetic constitution.

Cytological evidence for the occurrence of polyembryony in the avocado has been noted.* Only one specimen among hundreds examined contained within the ovule two embryo sacs (Figure 5*D*). Although presumably each of the two embryo sacs could have given rise to an embryo if fecundation had been completed only one embryo was formed. The other embryo sac had undergone degeneration.

In another specimen two developing

*SCHROEDER, C. A. The Ontogeny, Floral Anatomy and Embryology of the Avocado. (Unpub. doctoral thesis, Univ. of Calif., Los Angeles, 1943).

embryos were dissected out from a young fruit about 0.5 cm. long (Figure 5*E*). Both embryos were located near the micropyle and were closely appressed in a common chamber.

No evidence of nucellar embryony such as occurs in citrus and in the mango has been noted thus far in the avocado. All of the multiple embryos reported here apparently arise from gametic tissue. They may be "identical" or "fraternal," but they are not to be confused with "vegetatively reproduced" nucellar seedlings.

✦

WITH OUR FORE-BRAIN TIED BEHIND US

I AM just back from three days in Washington fighting through deferments for eighty-seven chemists, chemical engineers, bacteriologists, microanalysts and mycologists for the War Production Board's penicillin program. which includes twenty-one commercial producing plants and three university research programs. I am happy to say that at the moment it looks as though sixty-six of the needed men will be deferred. Deferment was refused for five of the men who are just about as necessary as any of the older ones because these five are only twenty-one years of age. Deferment of all but the 1944 class of technical students has now been canceled. This, with the attempt to put practically all men under twenty-six in the armed forces, means that this country is about to embark on one of the most remarkable experiments of all time.

This experiment means the practical elimination of technically trained men completing their courses between 1939 and 1948. During a highly technical war fought to lead up to an even more highly technical global peace and reconstruction such a cancelling out of nearly ten years of our new technical men comes about as near to an attempt at national suicide as could be thought up by the most ingenious of our enemies. Hitler's plan for the United States is supposed to include our reduction to an agricultural and mining unit to supply raw materials for the master race. I don't believe that Hitler is smart enough to think of such a perfect device to destroy our present and future technical manpower reserves.

Other countries are not so prodigal with their trained manpower. Recently, Dr. N. A. Ragozin, a petroleum expert on a Russian mission to this country, visited Penn State to look over our petroleum work. He brought with him an interpreter. After we had finished our business on petroleum, I asked the interpreter if he would ask the doctor what they were doing about technical students in Russia. He expressed some curiosity as to what I meant, and I explained that we were putting our technical students in the Army and Navy. He seemed rather astonished, and explained that Russia had more technical students in the universities now than ever before, and that they were not limiting them to girls, but were training as many girls and young men as were able to do technical work. He then told the interpreter to tell me that, when the Russians take back a city that has been destroyed by the Germans, one of the first things they do is to rebuild the local university and fill it with students.

In a recent address I mentioned 3000 chemists and chemical engineers who are in the armed forces and not engaged in technical work. My own personal guess is that the number is nearer 10,000 than 3000. Whatever the number, this is not the fault of the armed forces. Chemistry and chemical engineering are important in production. The Army and Navy are not producing units; they are consuming units. If a local board puts a chemist in the Army, it is practically impossible for the Army to find a job for him, because outside the Chemical Warfare Service, there are very few chemical jobs there. A great many of these chemists volunteered, thinking that the Army would find jobs for them, but, again, the Army has very few chemical jobs. The only thing you can profitably do with a misplaced chemist is take him out of the armed forces and put him in a gasoline refinery, rubber factory, a plant engaged in making explosives, or the like.

I fear very much that the fault is to a certain extent due ot the natural modesty of technical men and their absorption in their technical work. We have allowed the public to think that the achievements of chemistry and other sciences and technologies are pure magic. We see a wizard like Edison inventing things, and ignore the hundreds of young technicians in his laboratories. On that assumption, the public is perhaps justified in feeling that, for magic, all you need is one magician. Consequently, why should we not put all our young chemists and other technical men in the Army and leave a few old magicians to wave their magic wands in order to produce the thousands of materials needed to carry on a modern war? But the material for fighting this war is not produced by magicians, it must be made by trained technicians, and the time has come when technical men must overcome their distate for controversy and publicity and let the country know the truth.—Dean F. C. WHITMORE, *The Saturday Evening Post*, May 13, 1944.

THREE PEDIGREES OF MICROCEPHALY

With a Note on Their Genetic Implications

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THREE MICROCEPHALIC SIBLINGS
Figure 6

Three sisters, the last in a sibship of eight, and all microcephalic. As far as could be ascertained there were no predisposing causes, such as illness of the mother or x-ray therapy during pregnancy. The assumption that a recessive gene conditions the defect seems best to account for the observed incidence of this unfortunate deviation.

OUR present knowledge of the subject of microcephaly would suggest that many cases of congenital microcephaly are recessively determined, although the nature of the clinical syndrome associated with the gene mutation involved has yet to be accurately described.

A factorial analysis of sibships reported in the widely scattered literature (most of which is European) is, at most, suggestive and perhaps of general interest and should be interpreted with caution. Such an analysis is presented in Table 1. In it are included cases reported as "familial," "pin heads," "true" microcephalies, and "congenital microcephaly" in the absence of a history of X-ray irradiation, marked rachitis, or of a serious disease history in the etiology.

The difference between the number of pathotypes and expectancy is not sig-

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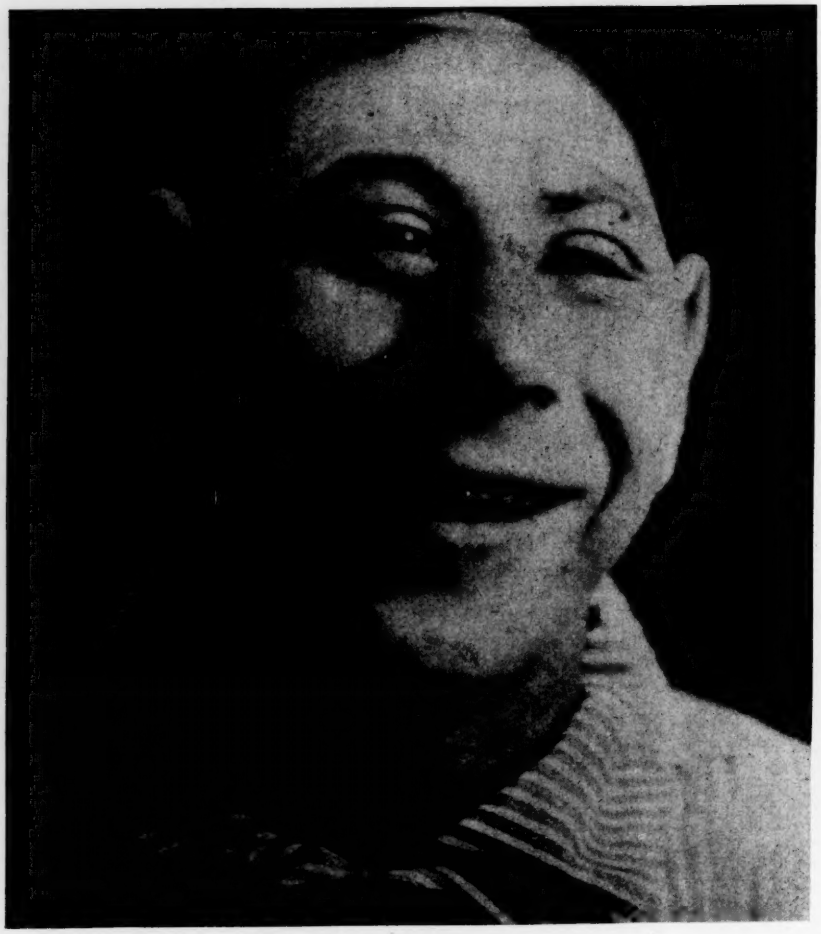


TWO OUT OF THIRTEEN

Figure 7

This boy's pedigree is shown in Figure 8, case 3. His mother was 33 when he was born. He has a microcephalic sister five years younger, with two intervening normal pregnancies.

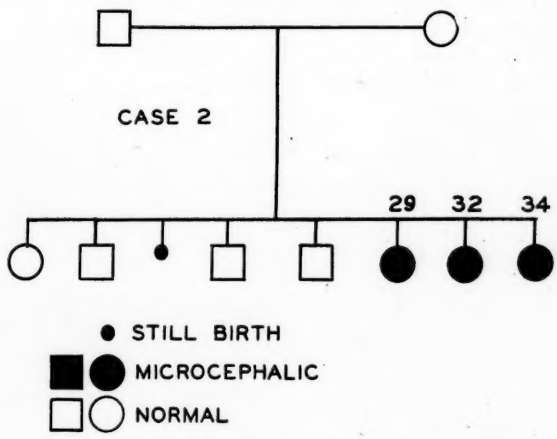
nificantly large. Similar results obtain when the analysis deals with pedigrees where at least two are affected: the number of affected is 33 and the expected number is 24.60; the standard error is 6.40.

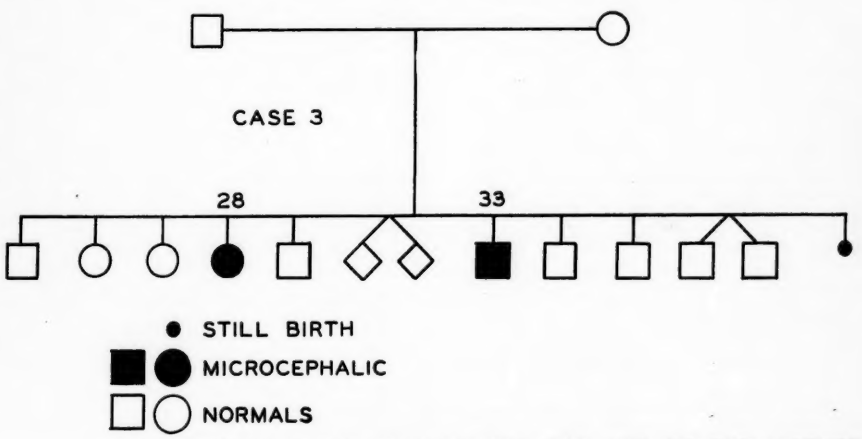
The data on consanguinity for the reported sibships cannot be determined with accuracy because the selection of cases may frequently have been biased. Nevertheless the evidence would seem to suggest a figure in excess of ten per cent.

A Report on Three Pedigrees of Microcephaly

Case 1

There were three affected in a sibship of five. The records for this case were destroyed during a fire which occurred


CASE 2
29 32 34
STILL BIRTH
MICROCEPHALIC
NORMAL
FIGURES 29, 32, AND 34 INDICATE AGE OF THE MOTHER AT THE TIME OF PREGNANCIES.



PEDIGREES OF MICROCEPHALY

Figure 8

In these pedigrees there was no record of relationship between the parents, so that consanguinity appeared not to be a factor in the outcropping of this recessive trait.

many years ago at the Institution. Dr. F. L. Keiser, the superintendent, examined the patients upon admittance. He reports that all three were typically microcephalic and that they toured with the Barnum Circus prior to commitment.

Case 2 (Figure 8)

The three patients in this family are girls, last born in successive pregnancies in a sibship of nine which includes two miscarriages. The mother was 29, 32, and 34 years of age at the time of the respective pregnancies.

The deliveries were uneventful. There is absence of a history of acute specific fever, of syphilis, or of X-ray irradiation during the pregnancies. In all three instances the abnormality was noted soon after birth. The early developmental histories were essentially negative, except for mental retardation. Walking and talking were delayed. The parents and other sibs are unaffected. The physical findings upon examination are not remarkable. One of the patients has in recent years developed a tubercular condition. Data on consanguinity are negative.

Case 3 (Figure 8)

The parents are unaffected and unrelated by blood. Both are mentally inferior but not defective. Data on pregnancy and birth are negative; the boy was born prematurely. X-ray irradiation was not employed during the pregnancies. There is no syphilitic history. The mother enjoyed relatively good health at the time of the births of the patients. She was 28 and 33 years of age at the respective pregnancies. The girl was fourth born and the boy eighth in a sibship of thirteen which includes one stillbirth.

Conclusions

The crux of the problem in microcephaly has yet to be solved: the determination of the clinical syndrome describable as genetically recessive. Nevertheless, the factorial analysis presented in this note would lend further credence to the suggestion of a few investigators, notably Dr. L. Penrose, that numerous cases of microcephaly are very likely recessively inherited.

It is suggested that parental consanguinity for cases of congenital microcephaly may well be in excess of 10%. No evidence for consanguinity is indicated for the three pedigrees described in this note.

Table I. Factorial Analysis of Sibships Containing Cases of Microcephaly

Size of sibship (s)	Number of sibships (n_s)	Number of pathotypes	Expected number $n_s \cdot \frac{p_s}{1-q^s}$	Variance $n_s s k_s$
1	2	2	2.00	0.00
2	6	6	6.86	0.73
3	8	9	10.38	2.10
4	9	12	13.17	3.78
5	6	12	9.83	3.55
6	5	8	9.12	3.88
7	2	4	4.04	1.94
8	5	9	11.11	5.86
9	3	10	7.30	4.17
10	2	6	5.30	3.18
11	2	2	5.74	3.61
12	3	4	9.29	6.06
13	-	-	-	-
14	2	5	7.43	4.89
	55	89	101.27	43.75
Difference 12.27				
Standard error 6.61				

✦

X-Chromosome Defects and Eunuchoidism

In each of the three families which showed an accumulation of primary gonadal deficiencies, eunuchoidism was found associated with color blindness, anosmia, synkinesia, and mental defect. The persistency of this association was regarded as evidence of a causal rather than a coincidental relationship.

Striking differences in the distribution of the various defects observed in eunuchoid kinships were taken as indicative of a particular genetic mechanism in the production of this as yet undescribed syndrome. They also supplied an opportunity to devise a generally applicable theory regarding the tendency of certain forms of hypogonadism and intersexuality to accumulate in individual families.

Color blindness occurred in both eunuchoid and non-eunuchoid members of the affected families, although it showed a tendency to associate itself with eunuchoidism only when hypogonadism appeared as a familial trait. Mental deficiency was also observed in eunuchoid as well as in non-eunuchoid individuals, but never without color blindness. Anosmia and synkinesia were not found in the absence of eunuchoidism, while eunuchoid defects occurred in the absence of either anomaly.

The most plausible genetic explanation for this association of gonadal and other developmental defects was seen in a *chromosomal aberration* involving breakage and partial translocation of the X-chromosome received by intersexes. Such an aberrant mechanism necessarily leads to a functional imbalance between the effects of the sex-controlling genes carried by the sex-chromosomes and the effects of the sex-controlling genes carried by the autosomes. — KALLMAN, F. J., W. A. SCHOENFELD and S. E. BARRERA. *Am. Jour. Ment. Def.* 48:203. 1944.

THE INHERITANCE OF NEVI

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ALMOST everyone has, or knows someone who has, a birthmark or nevus. It is surprising that not more work has been done on the inheritance of these, by no means, uncommon blemishes, and that there still exist many legends, beliefs and superstitions as to their cause. A great many people still attribute the appearance of nevi to that ancient superstition, "maternal impressions." They are alleged to owe their origin to an experience of the mother before the child was born. Such folk-fantasies tend to disappear with advance in knowledge but in backward areas they still persist. In southern Europe, for example, the average person attributes the presence of a nevus to the fact that the mother, during pregnancy craved some type of food. A brown nevus would suggest that there had been a craving for chocolates or other foods of that color. A red nevus is the product of an unsatisfied desire for steaks or strawberries.

The geneticist naturally can give no credence to these folk tales. Furthermore, there is considerable published evidence that nevi are inherited. In a study of 45 pairs of identical twins who possessed a total of 1,283 nevi, Siemens[20] found a correlation of $0.4 \pm .13$. Among 23 pairs of fraternal twins, there was a correlation of $0.2 \pm .19$. Meirowsky[11] and Scholl[17] working independently, found that there was a higher degree of correlation for number of birthmarks in monozygotic twins than for dizygotic twins. Meirowsky[11] reported a correlation of 0.78 for identical twins, 0.31 for fraternal twins.

In another study Brauer[1] found that 38 individuals in a total of 155 in a pedigree involving five generations possessed a nevus. Estabrook[2] in a study of five generations of one family found nine members in a total of 29 exhibiting a nevus. Meirowsky and Bruck[15] give a pedigree showing the inheritance of a nevus in which seven individuals out of 11 in three generations had the mark.

In view of the fact that there are few pedigrees in the literature these four accompanying family histories (set forth in Figure 9 and Table I) should be of interest.

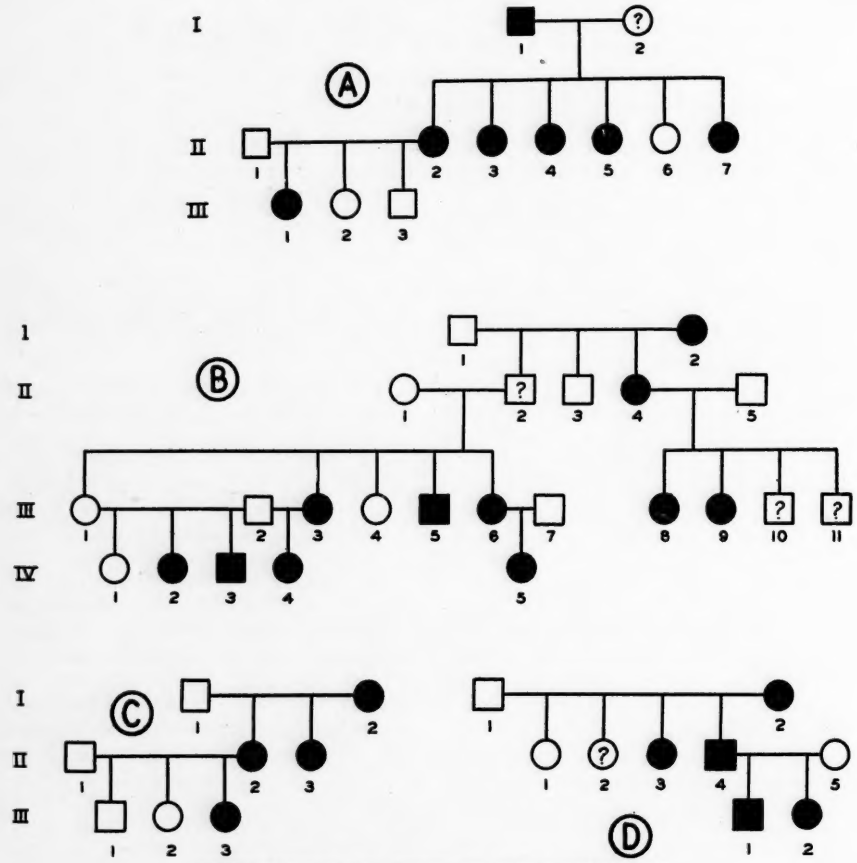
Of particular interest is the tendency illustrated in Table I, for the same kind of nevus to occur in the same position among relatives. This inherited localization does not occur universally but its frequency is very high, not only in these pedigrees but in those previously reported by others.

In Pedigree *A*, the same type and size of nevus was inherited on the face by two members and on the back by three other members. In Pedigree *B*, six members possessed the same type of nevus on the face, two members possessed a nevus on the back and four individuals showed the nevus somewhere on the left leg. Perhaps the most striking case is shown in Pedigree *C*. Here two large brown nevi (which were side by side) were inherited from grandmother to two daughters, to granddaughter, in the same place—on left side of body at the level of the waist. In another family (Pedigree *E*) not here recorded in detail, the author found two daughters exhibiting a nevus on the chin in the exact position in which a nevus occurs on the father. The nevi differed only in size and in number of hairs, the father's being larger and more hairy. In three other families, where the pedigrees of the family were impossible to trace, the author found that in:

Pedigree F—brother and sister had a brown nevus on chest

Pedigree G—father and daughter had a dark brown nevus on back

The author wishes to acknowledge the assistance and guidance of Dr. P. C. Mangelsdorf in the preparation of this study



INHERITANCE OF NEVI IN FOUR FAMILIES

Figure 9

The location and description of the nevi is given in Table I. There is a remarkable consistency in type of nevus and in location in these families, and in others observed by the author and reported elsewhere in the literature.

Pedigree H—mother and daughter had a large dark nevus below the chest

Meirowsky[13] found a pigmented mole in the ileocecal region of the mother and another very similar one on the corresponding place in her three-year-old son.

Estabrook[2] noted a dark brown nevus occurring in the same region of the body (abdomen and navel region) in four members in two generations of a family. Two other members of this family inherited a nevus in corresponding regions of the neck.

Leven[7] described a case where the father showed a nevus and his two daughters did not possess the anomaly. However, in the F_2 generation, two children showed the nevus, the same size and location as the grandfather. In a later study, Leven[8] described a family where the grandmother and three grandchildren showed the same *Nevus pigmentosus* of the same size in the same position.

In Brauer's study[1] the inheritance of the localization of the nevus is clearly

shown. Brauer states that the nevus occurred in the supra-orbital region between the outer corner of the eye and the hair margin in 38 out of 155 members in five generations of a family. There was variation in number, size and shape of the nevi and in the same individual the two sides were not affected alike. In two individuals in this family, the nevi were asymmetrical, occurring on only one side of the head, while among their descendants, the characteristic symmetry reappeared. This suggests that in these two exceptions, modifying factors or other conditions suppressed the appearance of the characteristic nevi; nevertheless, the nevi were transmitted to the progeny.

TABLE I—Description of Nevi

Individual	Description of nevus	Localization
	Pedigree A	
I-2	light brown, raised, about 6 cm. in diam.	face
II-2	ditto	back
II-3	ditto	back
II-4	ditto	chest
II-5	ditto	face
II-7	ditto	back
III-1	ditto	right ear
	Pedigree B	
I-2	hairy mole, about 4 cm. in diam.	face
II-4	ditto	face
III-3	large hairy mole and three smaller ones	back
III-5	white nevus about the size of a penny	face
III-6	large dark brown hairy nevus	face
III-8	brown hairy mole	left leg
III-9	ditto	chin
IV-2	large "strawberry" nevus	left leg
IV-3	large white nevus	left ankle
IV-4	large brown hairy nevus	left knee
IV-5	two brown hairy moles	back and face
	Pedigree C	
I-2	two brown nevi side by side	left side of waist
II-2	ditto	ditto
II-3	ditto	ditto
III-3	ditto	ditto
	Pedigree D	
1-2	numerous light brown, raised nevi	all over body
I1-3	ditto	ditto
I1-4	ditto	ditto
III-1	numerous dark brown, raised nevi	ditto
III-2	ditto	ditto

The results of my studies considered in conjunction with the previously reported studies of Siemens,[18,19,20] Meirowsky,[10,11,12,13,14] Brauer[1] and Estabrook[2] indicate that the inheritance of nevi involves dominant or partly dominant genes. These genes may be entirely suppressed in certain generations of a pedigree and thus the anomaly may not appear in a generation. However, the anomaly may reappear in a later generation. Estabrook[2] in his study of five generations found a case where neither parent possessed a nevus (though grandfather had a nevus) and produced four children, two of which had a birthmark. The characteristic localization of the nevi in different families suggests that different genes are involved in different families or that different complexes of modifying factors are involved.

Nevi are found to be inherited in approximately the same manner as neoplasms (cf. Macklin[9]). The frequency of nevi and neoplasms tend to be about the same in a pedigree, the type of nevus and neoplasm, and their localization tend to be inherited. Whether there is any connection between the inheritance of nevi and neoplasms can not be determined until more extensive data can be collected. There are many cases cited in published literature where a nevus, especially the smooth, hairless variety, developed into a cancerous growth. It may be of interest to note that in Pedigree *B*, No. 1 died of cancer of the breast at the age of 30, No. 3, and No. 6, had a growth, possibly precancerous, removed from the breast at the ages of 27 and 28 respectively. This tendency for the same type of nevus and neoplasm to be inherited in the same location suggests that the mode of the inheritance may be quite similar in the two cases.

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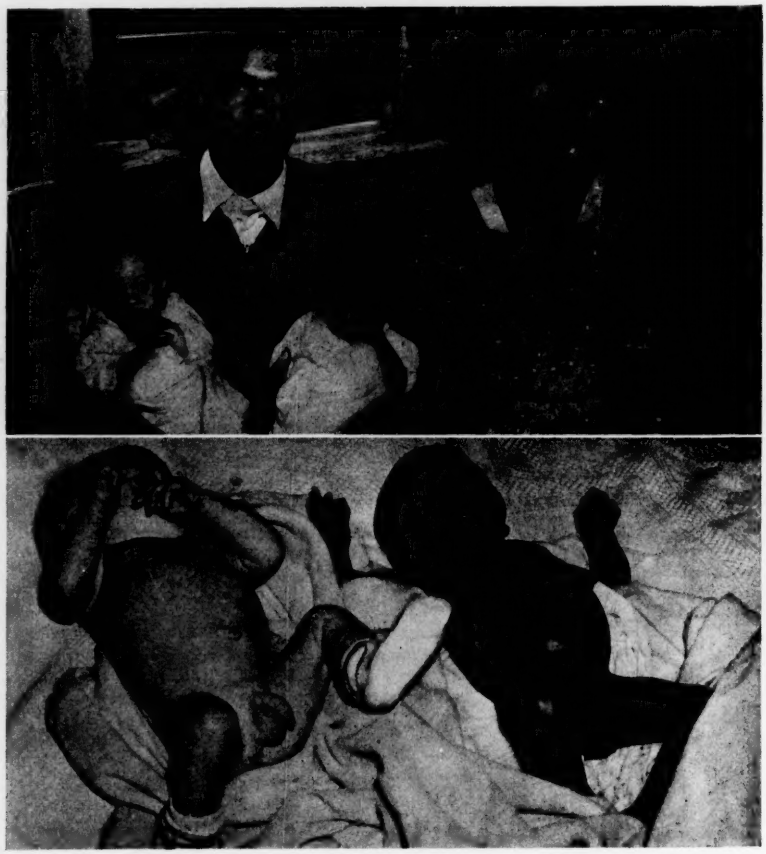

STRONG TWINS

Recessive albinism outcrops in progeny of normal parents.

LETHAL AND SUBLETHAL CHARACTERS IN FARM ANIMALS

A Check-List and Proposed Numbering System

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IN 1937 Eaton presented in this JOURNAL (28:320) a summary of lethal conditions in domestic and laboratory animals and in man. In the six years that have elapsed, a number of new lethal and sub-lethal characters have been described, particularly in farm animals, including eleven in cattle, three in horses, three in swine, one in sheep, ten in chickens and two in turkeys. It seems likely that with the numerous studies on inbreeding which are being currently carried on in these animals, further discoveries of lethals are imminent. It may therefore be desirable to bring Eaton's summary up to date at least with regard to the above-mentioned species. A system of numbering which would facilitate future reference may also have its advantages and is presented in the following list. The arrangement of the characters is by species, and within species in chronological order of the first reference to the particular condition. There are, undoubtedly, some oversights and omissions, but since considerations of priority are negligible in this connection, they may be remedied eventually without changing the numbering system. For instance, character A13 may appear misplaced, so far as the chronological order is concerned, yet as may be seen by reference to Eaton's list a mention of the particular condition of ankylosis has made been by the same author (Stang) as early as 1935.

It may also be noted that several characters listed by Eaton have been omitted from the present list. They represent conditions whose inclusion, in the opinion of the author, is not justified because the evidence, either of the hereditary or the lethal nature of the defects, is unsatisfactory. The characters are in the order of their appearance in Eaton's summary as follows:

A) Cattle—
Congenital ichtyosis,
foetal resorption.

C) Swine—
Excessive fatness,
foetal mortality,
hypotrichosis.

E) Chickens—
Embryonic lethals,
malposition of embryos.

It is possible that further investigation may serve to reinstate these conditions in a later revision of such a list.

All of the characters, unless otherwise noted, are recessive. Extensive use of both Eaton's summary and that of Hutt (1934 CV 24:1) has been made in this compilation. The publications cited by initial are listed at the end of the article.

A. Cattle

Number	*Character*	*Comments*	*Investigators*
A1	Achondroplasia	Endocrine defect affecting skeleton. Heterozygotes exhibit achondroplasia.	Seligmann, 1904 JPB 9:311. Wilson, 1909 RDS 12:1. Crew, 1923 RS 95:228. Crew. 1924 RSM 17:39. Punnett, 1936 JG 32:65.
A2	Epitheliogenesis imperfecta	Denudation of skin facilitating bacterial invasion, which leads to septicaemia.	Cole, 1919 JH 10:303. Hadley and Cole, 1923 WRB 86. Hadley, 1927 JH 18:487. Hadley and Warwick, 1927 JAVMA 70:492. Regan, Mead and Gregory, 1935 JH 26:357. Wipprecht and Horlacher, 1935 JH 26:363.

Number	*Character*	*Comments*	*Investigators*
A3	Achondroplasia	Milder form than exhibited in dominant achondroplasia; affected calves born alive. The cases described by Gregory *et al* are phenotypically distinct from the others.	Wriedt, 1925 ZTZ 3:223. Mohr, 1926 ZiAV 41:59. Weinkopf, 1927 BTW 43:62. Wriedt, 1930 HL. Carmichael, 1933 N 131:878. Punnett, 1936 JG 32:65. Brandt, 1941 JH 32:183. Gregory, Mead and Regan, 1942 JH 33:317.
A4	Hypotrichosis congenita	Hairlessness; affected calves die shortly after birth.	Mohr and Wriedt, 1928 JG 19: 315. Wriedt, 1930 HL. Butz, 1934 DTW 42:653. Eisele, 1936 Z 11:432. Johansson, 1941 IGCE :169.
A5	Acroteriasis congenita	Appendages partly absent — "amputated"; jaws deformed; affected calves often stillborn.	Wriedt and Mohr, 1928 JG 20: 187. Wriedt, 1930 HL.
A6	Mummification	Stiffened legs, shortened neck, many foetuses mummified at term.	Loje, 1930 TL 10:517.
A7	Lameness	Affected calves born alive, but lame in hind legs.	Loje, 1930 TL 10:517.
A8	Muscle contracture	Head, neck and limb muscles affected; affected calves may be born alive.	Mohr, 1930 NV 14:1. Hutt, 1934 JH 25:41.
A9	Ankylosis of jaw	Articulation of lower jaw ossified.	Mohr, 1930 NV 14:1.
A10	Short spine	Fusion of vertebrae resulting in shortened spine; affected calves stillborn or die at birth.	Mohr and Wriedt, 1930 JG 22: 279.
A11	Ljutikow's lethal	Inferred from records of stillborn calves. Contrary to description in other check-lists, Ljutikow finds no recorded evidence of abnormalities in the stillborns.	Ljutikow, 1932 BZ 1:21.
A12	Congenital dropsy	Accumulation of water in body cavity and subcutaneously; affected calves born alive.	Larsson, 1935 LVH :310. Johansson, 1941 IGCE :169.
A13	General ankylosis	All joints ossified; cleft palate.	Stang, 1940 ZZ 46:280.
A14	Impacted molars	Premolars impacted; "parrot-mouth" appearance; affected calves alive at full term.	Heizer and Hervey, 1937 JH 28:123.
A15	Achondroplastic micromelia	Calves cannot survive on pasture because of imperfection of lower jaw in addition to shortened limbs.	Ljutikow, 1937 BZ 6:413.
A16	Atresia ani	Imperforate anus; affected calves do not survive surgical treatment.	Kuppuswami, 1937 IJ 7:305.
A17	Deformed limbs	Crooked legs, sometimes with ankylosis. Calves stillborn or if alive, unable to stand.	Ruzhevsky, 1938 BZ 7:547.
A18	Brain hernia	Opening in skull involving frontal and parietal bones, affected calves either born alive or stillborn.	Shaw, 1938 JH 29:319.
A19	Agnathia	Lower jaw half the length of normal; affected calves unable to nurse.	Annett, 1939 JG 37:301.

Number	*Character*	*Comments*	*Investigators*
A20	Agnathia	Jawless, accompanied by wattle-like structure. All cases studied were foetal.	Ely, Hull and Morrison, 1939 JH 30:105.
A21	Sex-linked lethal	Evidence of existence based on abnormal sex ratios (shortage of males).	Andreesen, 1940 ABA 10:24.
A22	Abnormal skull	Nasal openings fused. Die at birth or shortly after. Possible inhibitor of lethal expression also found.	Ilancic, 1940 Z 15:129.
A23	Missing phalanges	First and second phalangeal bones missing, otherwise calves born at full term are normal.	Johansson, 1941 IGCE :169.
A24	Hydrocephalus	Internal hydrocephalus accompanied by bone abnormalities.	Cole and Moore, 1942 JAR 65: 483.
A25	Congenital spasm	Vertical intermittent spasms of head and neck. Calves die shortly after birth.	Gregory, Mead and Regan, 1944 JH 35:195.

B. Horses

B1	Atresia coli	Closure of colon, often associated with brain defects; colts born alive.	Nusshag, 1925 BTW 41:646. Yamane, 1925 FEATM :815. Yamane, 1927, ZiAV 46:188.
B2	Fredriksborg lethal	Evidence for existence inferential; high degree of sterility in inbred matings of white horses led to the postulation of existence of a lethal factor.	Wriedt, 1924 ZTZ 1:231; 1930 HL.
B3	Sex-linked lethal	Evidence of existence based on abnormal sex ratios (shortage of males).	Kislovsky, 1932 ZTZ 24:269.
B4	Epitheliogenesis imperfecta	Denuded skin on limbs, occasionally missing hoof. Born alive but none of 5 observed colts survived. Evidence of hereditary nature of defect inferential.	Plank, 1936 NFTA :233.
B5	Deformed forelimbs	Atrophied muscles or lack of balance between tendons.	Prawochenski, 1936 JH 27:411.
B6	Umbilical hernia	No information given by author.	Prawochenski, 1941 IGCE :241.
B7	Absence of eye orbits	No information given by author.	Prawochenski, 1941 IGCE :241.

C. Swine

C1	Brain hernia	Skull opening involving frontal and parietal bones, affected piglings born alive.	Goldsmith, 1922 JH 13:69. Nordby, 1929 JH 20:229. Hughes and Hart, 1934 JH 25: 111.
C2	Paralysis	Hind limbs paralyzed; affected piglings born alive.	Mohr, 1930 NV 14:1.
C3	Atresia ani	Closure or diverticulum of anal opening in piglings born alive; mode of inheritance not clear.	Kinzelbach, 1931 ZiAV 60:84. Carstens, Wenzler and Dürr, 1937 Z 12:205. Berge, 1941 JH 32:271.
C4	Cleft palate	Affected piglings born alive but unable to nurse; line died out before genetic nature of this condition was definitely established.	McPhee, Russell and Zeller, 1931 JH 22:393. Koch and Neumüller, 1932 DTW 40:353.

	Character	*Comments*	*Investigators*
C5	Thickened fore-limbs	Muscle fibers displaced by connective tissue infiltration; affected piglings born alive.	Walther, Prüfer and Carstens, 1932 Zr 7:178. Carstens, Wenzler and Dürr, 1937 Z 12:205.
C6	Muscle contracture	Rigid fore-limbs; affected piglings usually stillborn.	Hallquist, 1933 H 18:219.
C7	Split ears	Associated with deformed hindquarters and sometimes cleft palate; affected piglings usually born dead.	Annett, 1938 JH 29:469.
C8	Hydrocephalus	Fluid found in subarachnoid spaces; affected piglings sometimes stillborn.	Blunn and Hughes, 1938 JH 29: 203.
C9	Amputated	Both front and hind appendages completely missing, though shoulder blades and pelvic bones present; affected piglings born alive.	Johnson, 1940 JH 31:239.
		D. Sheep	
D1	Muscle contracture	Flexure of limbs and wry neck; affected lambs stillborn.	Roberts, 1926 JMA 33:795. Roberts, 1929 JG 21:57.
D2	Earless and cleft palate	Often associated with tripartite claws; apparently, affected lambs are born alive (listed as "sublethal" by Mohr).	Mohr, 1929 Z 4:105.
D3	Paralysis	Hind limbs paralyzed; affected lambs born alive.	Zophoniasson, 1929 NJ 11:327.
D4	Rigid fetlocks	Skeleton, including skull, deformed and associated with short wool and a hernia; affected lambs born alive.	Zophoniasson, 1929 NJ 11:327.
D5	Amputated	Limbs amputated at fetlocks; mode of inheritance not clear.	Kroon and Planck, 1931 TD 58: 681. Kroon and Planck, 1932 BG 8: 213. Höhn, 1942 DTW 50:349.
D6	Lethal gray	2 gray to 1 black ratio observed in matings between gray parents. No homozygous gray adults found.	Constantinescu, 1932 INZR 1:15.
		3 gray to 1 black observed in matings between gray parents, but no homozygous gray adults found. Presumed homozygotes die in early postnatal life; digestive disturbances associated with cause of death.	Glembotsky, 1935 PZ (2):41. Contescu and Epureanu, 1939 ZTZ 44:211.
D7	Dwarf	Thyroid disturbance leading to death within a month after birth.	Bogart and Dyer, 1942 JAS 1:87.
		E. Chickens	
E1	Creeper	Shortened extremities in heterozygotes. Homozygotes usually die in first week of incubation.	Cutler, 1925 JH 16:353. Landauer, 1941 SB 236 contains bibliography of numerous papers. Hamburger, 1942 BS 6:311.
E2	Congenital loco	Affected chicks unable to stand, head bent backwards.	Knowlton, 1929 OB :253.
E3	Sticky	Lack of absorption of amniotic and allantoic fluids; bones deficient in calcium; affected embryos die in last week of incubation.	Byerly and Jull, 1932 JEZ 62: 489.

E4	Wyandotte lethal	Linked with recessive white in the Wyandotte breed.	Dunn, 1932 AN 57:345.
E5	Congenital palsy	Tremor affecting hatched chicks; occasionally affected chicks survive.	Hutt, 1932 IGCI-2:96. Hutt and Child, 1934 JH 25:341.
E6	Cornish lethal	Shortened extremities in heterozygotes. Homozygotes die in last week of incubation.	Landauer, 1935 JG 31:237.
E7	Sex-linked lethal	Evidence of existence based on abnormal sex ratios (shortage of females).	Upp and Waters, 1935 PS 14: 372.
E8	Amaxilla	Maxillae absent or reduced, beak bent to one side; most of the affected chicks unable to hatch.	Asmundson, 1936 JH 27:401.
E9	Flightless	Defective structure of flight feathers. No homozygotes raised to maturity.	Warren, 1937 JH 28:17.
E10	Malformed skeleton	Curvature of spine and pelvis; birds developing the defect are unable to move. Several recessive genes postulated, but data given inadequate to formulate any definite conclusions.	Czaja, 1939 WPCC :55.
E11	Naked	Affects feather follicles, reducing down and plumage; sex-linked; half of the affected chicks hatch, but only half of these survive.	Hutt and Sturkie, 1938 JH 29: 371.
E12	Short beak	Upper mandible and certain long bones shortened. 87% of homozygotes die before hatching.	Landauer, 1941 G 26:426.
E13	Microphthalmia	Diameter of eyeballs reduced; mortality occurs in late embryonic or early post-hatching stages.	Jeffrey, 1941 JH 32:310. Gruenwald, 1944 AR 88:67.
E14	Micromelia	Two genes involved; double recessives exhibit "parrot-beak" and shortened and thickened extremities.	Asmundson, 1942 JH 33:328.
E15	Talpid	Affected embryos exhibit extra digits in fore and hind limbs, frequently webbed; ectopia always present; homozygotes usually die at 8-10 days of incubation.	Cole, 1942 JH 22:83.
E16	Chondrodystrophy	Extreme type of bone modification; affected embryos fail to hatch.	Lamoreux, 1942 JH 33:275.
E17	Deformed mandibles	Lower mandible missing, upper deformed; surviving homozygotes unable to pip shell; cerebral hernia present.	Marble, Harper and Hammers, 1944 PS 23:114.
E18	Wingless	Wings absent or vestigial, affected embryos do not survive.	Waters and Bywaters, 1943 JH 34:213.
E19	"Chondrodystrophy"	Shortened bones of limbs, but no parrot beak; few homozygotes hatch, none surviving beyond 7 days after hatching.	Hays, 1944 AN 78:54.

F. Turkeys

F1	Albinism	Sex-linked recessive; only about a quarter of carrier females hatch; most of these do not survive for long, largely because of blindness associated with the absence of pigment.	Hutt and Mueller, 1942 JH 33: 69.
F2	Short spine	Shortened neck and body, affected embryos fail to hatch.	Asmundson, 1942 SEBM 50:120.

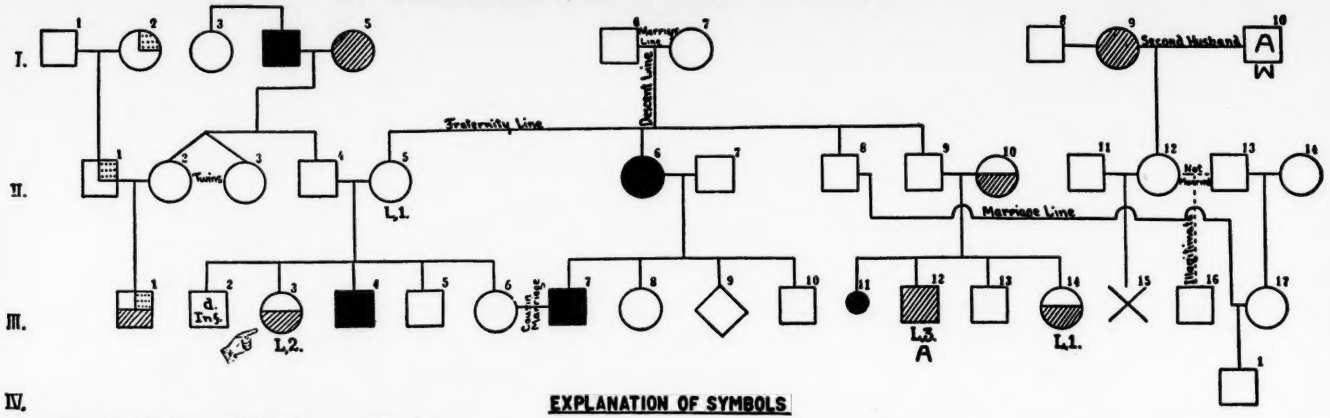
G. Ducks

G1	Crest	Heterozygotes crested; homozygotes have cerebral hernia and do not hatch.	Rüst, 1932 AG 6:110.

List of Abbreviations in Literature References

ABA—*Anim. Breed. Abstr.*
AG—*Arch. Geflügelk.*
AN—*Am. Naturalist.*
AR—*Anat. Rec.*
BG—*Biol. Generalis.*
BS—*Biol. Symposia.*
BTW—*Berlin. Tierärztl. Wochenschr.*
BZ—*Biol. Zhurn.*
CV—*Cornell Vet.*
DTW—*Deutsch. Tierärztl. Wochenschr.*
FEATM—Trans. Sixth Congr. Far East. Assoc. Trop. Med.
G—*Genetics.*
H—*Hereditas.*
HL—Heredity in Livestock, Macmillan, London.
IGCE—Proc. Seventh Int. Genet. Congr., Edinburgh.
IGCI—Proc. Sixth Int. Congr. Genet., Ithaca.
IJ—*Indian J. Vet. Sci. Anim. Husb.*
INZR—*Ann. Inst. Nat. Zootech. Roumanie.*
JAR—*J. Agr. Res.*
JAS—*J. Anim. Sci.*
JAVMA—*J. Am. Vet. Med. Assoc.*
JEZ—*J. Exp. Zool.*
JG—*J. Genet.*
JH—*J. Hered.*
JMA—*J. Ministry Agr.*
JPB—*J. Path. Bact.*
LVH—*Lantbr. Veck. Handl.*
N—*Nature.*
NFTA—*Nene Forsch. Tierz. Abstammungsl.*
NJ—*Nord. Jordbrugsf.*
NV—*Naturens Verden.*
OB—*Ore. Agr. Exp. St. Bull.*
PS—*Poultry Sci.*
PZ—*Probl. Zhivotn.*
RDS—*Proc. Roy. Dublin Soc., N. S.*
RS—*Proc. Roy. Soc. B.*
RSM—*Proc. Roy. Soc. Med.*
SB—*Storrs Agr. Exp. St. Bull.*
SEBM—*Proc. Soc. Exp. Biol. Med.*
TD—*Tijdschr. Diergenesek.*
TL—*Tidsskr. Landok.*
WPCC—Proc. Seven World's Poultry Congr., Cleveland.
WRB—*Wis. Agr. Exp. St. Res. Bull.*
Z—*Züchtungsk.*
ZiAV—*Zeit. ind. Abst. Vererb.*
Zr—*Züchter.*
ZTZ—*Zeit. Tierz. Züchtungsbiol.*
ZZ—*Zeit. Zücht. B.*

SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a). Standard for all Pedigree Charts:

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d.Inf.=Died in Infancy; ☞ Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III.7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic; F, feebleminded; I, insane; M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b). Especially devised or selected for this particular pedigree-chart:

■ ● = Successful leaders in politics. ◔ ◔ = Extra thumb on right hand. L.1.=Highly successful author. L.3.=Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from A.G.A.

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